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# Summary

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## Scope

At request of the Minister of Social Affairs and Employment, The Health Council of the Netherlands sets health-based recommended occupational exposure limits (HBR-OEL) for toxic substances in the workplace air. These recommendations are made by the Council's Dutch Expert Committee on Occupational Standards (DECOS). It constitutes the first step in a three step procedure which leads to legally binding occupational exposure limits.

The present report on hydrogen sulphide was prepared in cooperation with the Nordic Expert Group (NEG). The joint report on the consequences of occupational exposure to hydrogen sulphide, published in Sweden in 2001 (Arbete och Hälsa 2001:14), is included in part two of this document. Part 1 mainly consists of a summary of the most important health effects and a health hazard assessment by DECOS.

The committee's conclusions are based on scientific publications obtained from data retrieval systems from prior to August 2004.

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## Physical and chemical properties

H<sub>2</sub>S is a colourless gas (CAS number 7783-06-4) with a strong odour of "rotten eggs" (odour threshold 0.13 ppm, 0.18 mg/m<sup>3</sup>). Its melting point is -85.5°C and the boiling point is -60.7°C. Solubility in water and ether is 0.4 and 2.1% (w/w),

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respectively. Hydrogen sulphide has a relative density of 1.19 and a vapour pressure of 2026 kPa. The substance is flammable and explosive in air and may even be ignited by static discharge.

Large quantities of H<sub>2</sub>S are used in the production of deuterated water. Hydrogen sulphide is formed in manufacturing processes whenever elemental sulphur or sulphur compounds are present with organic compounds at high temperatures. Examples of industries where H<sub>2</sub>S can be generated include petrochemical plants, coke oven plants, viscose rayon industries and tanneries.

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### Monitoring

The main sampling method is sampling on filters impregnated with silver nitrate. The resulting silver sulphide is dissolved in alkaline cyanide solution and analyzed for sulphide by differential pulse polarography (detection limit is 0.6 mg/m<sup>3</sup>). H<sub>2</sub>S can also be analysed by gaschromatography (with flame ionization detection or a flame photometric detection) on the spot or after collection in plastic laminate bags. Several other reading methods are available.

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### Current limit values

In the Netherlands, the present MAC-value (maximal allowed concentration) is 10 ppm (14 mg/m<sup>3</sup>). In Denmark, Finland, Germany, Iceland, Norway, the USA (ACGIH\*) 10 ppm (14 mg/m<sup>3</sup>) is recommended as occupational exposure limit. In Finland in addition a 15 minutes value of 21 mg/m<sup>3</sup> is available. The United Kingdom derived and OEL (twa 8 hours) of 5 ppm (7 mg/m<sup>3</sup>) and a STEL \*\* (15 minutes) of 10 ppm (14 mg/m<sup>3</sup>). Ceiling values of 15 ppm (21 mg/m<sup>3</sup>) are found in Iceland and Sweden. In the USA a ceiling value of 10 ppm (14 mg/m<sup>3</sup>, NIOSH\*\*\*), 20 ppm (28 mg/m<sup>3</sup>, OSHA \*\*\*\*) and a 10 minutes peak value of 50 ppm (70 mg/m<sup>3</sup>, OSHA) are recommended.

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\* American Conference of Governmental Industrial Hygienists (ACGIH)  
\*\* Short Term Exposure Limit (STEL)  
\*\*\* National Institute for Occupational Safety and Health  
\*\*\*\* Occupational Safety and Health Administration (OSHA)

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## Kinetics

Uptake of H<sub>2</sub>S is mainly via the inhalatory route. After exposure of mice and rats increased sulphide concentrations were shown in the lungs, liver, kidney, olfactory epithelium and the brain. H<sub>2</sub>S is mainly metabolised mainly by the liver to (thio)sulphate and excreted via urine. Due to rapid metabolism there is no bioaccumulation of sulphide.

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## Effects

### Human data

H<sub>2</sub>S is a gas which seems to cause irritation of the eyes. In viscose rayon workers effects on eyes were observed at levels lower than 20 ppm (28 mg/m<sup>3</sup>), however, in these industries simultaneous exposure to CS<sub>2</sub> was measured as well.

Short term occupational exposure might lead to lung function impairment and neurobehavioral changes. Data are, however, limited. Acute effects after exposure to high concentrations include pulmonary oedema (at ca. 700 mg/m<sup>3</sup> and above) and “knock down”. In asthmatics, no significant effects on the airway resistance were found after exposure to 2.8 mg/m<sup>3</sup> (2 ppm) H<sub>2</sub>S for 30 minutes. Exposure to 14 mg/m<sup>3</sup> (10 ppm) H<sub>2</sub>S for 15 minutes caused no significant changes in pulmonary function in human volunteers. However, they were only exposed through the mouth.

In pulp-mill workers, an excess mortality from cardiovascular disease (and coronary heart disease) has been observed after exposure to H<sub>2</sub>S and organic sulphur compounds, however exposure measurements were not performed. Other effects reported after prolonged exposure include olfactory fatigue (>100 ppm or 140 mg/m<sup>3</sup>).

### Animal studies

In laboratory animals, exposure to 140-420 mg/m<sup>3</sup> H<sub>2</sub>S (100-300 ppm for a few hours) leads to irritation of the eyes and the mucous membranes of throat and nasal cavity. The LC<sub>50</sub> (ie. concentration at which 50% of the animals died) in rats after inhalation exposure (for four hours) was 617-691 mg/m<sup>3</sup> (~500 ppm). Effects seen after short exposure to high concentrations were cytotoxic lesions in the lungs and pulmonary oedema. Neurotransmitter levels in the respiratory centers in the brainstem were increased as well.

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Rodents exposed to H<sub>2</sub>S at 25-100 ppm (35-140 mg/m<sup>3</sup>) showed inhibition of cerebral cytochrome oxidase activity, increased L-glutamate levels in the hippocampus and EEG changes, lesions of the olfactory mucosa, various cardiac arrhythmias and an increased number of reticulocytes. Additional studies in rats on behavioural effects (motor activity, learning) showed an increased latency time in the (re)acquisition of performance in mazes at concentrations of 175 mg/m<sup>3</sup> and above and decreased motoractivity at 112 mg/m<sup>3</sup> and above.

Effects on the olfactory mucosa became apparent in rats sub-chronically (70-90 days) exposed to 30 ppm H<sub>2</sub>S (42 mg/m<sup>3</sup>). The effects reported included olfactory neuronal loss, basal cell hyperplasia of the mucosal lining. In addition, bronchial epithelial hypertrophy and hyperplasia was observed after exposure to H<sub>2</sub>S for 90 days. The NOAEL was 14 mg/m<sup>3</sup>.

No genotoxic effects were reported. Toxicity due to long-term exposure was not investigated.

No effects on reproduction and development were reported in rats exposed to H<sub>2</sub>S (14, 42 and 112 mg/m<sup>3</sup>) during mating, gestation and lactation. In the same study no effects on growth, development and behaviour of the pups were found. No gross or microscopic abnormalities were observed in the central nervous system of the offspring.

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### **Hazard Assessment and recommended occupational exposure limit.**

There is limited information concerning the effects of H<sub>2</sub>S after acute exposure. Only a few cases have been described in which acute exposure (to concentrations exceeding 1400 mg/m<sup>3</sup>) caused a cessation of respiration. In asthmatics, exposure to 2.8 mg/m<sup>3</sup> H<sub>2</sub>S for 30 minutes did not result in statistically significant respiratory effects. Mouth only exposure for 15 minutes (to 14 mg/m<sup>3</sup>) did not cause significant changes in pulmonary functions.

Acute or short-term exposure to H<sub>2</sub>S, resulted in experimental animals to inhibition of cytochrome oxidase in the lung cells, and local irritation of eyes and throat.

The committee is of the opinion that the data concerning acute of short term exposure show that a short term exposure limit is not indicated.

There is limited human information concerning the health effects after prolonged exposure to H<sub>2</sub>S as well. Exposure to 1-5.6 mg/m<sup>3</sup> H<sub>2</sub>S caused eye irritation in viscose rayon workers. However, eye irritation in these industries might be a result of combined exposure with CS<sub>2</sub>. There are no data concerning the effects of H<sub>2</sub>S alone below levels of 28 mg/m<sup>3</sup>.

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In rats, exposure to H<sub>2</sub>S causes nasal lesions (olfactory neuron loss and basal cell hyperplasia) and bronchial epithelial hypertrophy and hyperplasia after exposure to H<sub>2</sub>S for 70-90 days (6 hours/day, 7 days/week). The NOAEL (no observed adverse effect level) for these effects is 14 mg/m<sup>3</sup>. The committee is of the opinion that these are the critical effect. For the establishment of the health based occupational exposure limit (HBROEL) to committee takes several aspect into account. As default, the committee usually compensates for differences between rats and man. However, as the critical effect is local, the committee did not find such factor necessary. For the differences in exposure pattern (sub-chronic in the experimental setting versus the chronic occupational exposure) and the limited data concerning the pathology, the committee uses a factor 2. Finally, to compensate for interindividual differences the committee uses a factor 3 as well.

Considering all these aspects, starting from a NOAEL of 14 mg/m<sup>3</sup> and using an extrapolation factor of 6 the committee recommends an HBROEL twa 8 hours for H<sub>2</sub>S of 2.3 mg/m<sup>3</sup> (~1.6 ppm).

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### **Health based recommended occupational exposure limit**

DECOS recommends an HBROEL twa 8 hours for H<sub>2</sub>S of 2.3 mg/m<sup>3</sup> (~1.6 ppm).